

# Intraoperative Assessment of Cerebral Activity During Open Heart Surgery

William B. Shaffer, C.C.P.  
Chief Certified Clinical Perfusionist

Dorothy F. Farrell, C.C.P.  
Certified Clinical Perfusionist

Paul G. Barash, M.D.  
Assistant Professor of Anesthesiology

Judith M. Gatehouse, C.C.P.  
Certified Clinical Perfusionist

Susan W. Iverson, B.A., C.P.  
Clinical Perfusionist

Departments of Surgery and Anesthesiology  
Yale University School of Medicine  
333 Cedar Street  
New Haven, Connecticut 06510

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## INTRODUCTION

During cardiopulmonary bypass (CPB), disturbances in neurological status may be reflected by changes in cerebral activity. The etiology of this dysfunction is thought to be micro-emboli (air or particulate matter) decreases in perfusion pressure, blood flow, and hypoxia.<sup>1,2</sup> Other factors to be considered in relation to the incidence of neurological damage include: patient's age, the duration of cardiopulmonary bypass, the pre-operative physiologic and neurologic status, nature of the surgery, history of previous cardiac surgery, type of oxygenator and the priming solution used.<sup>3</sup> Previously, electroencephalograms (EEG) were used to monitor cerebral activity and deemed a highly accurate method of diagnosing intra-operative neurological deficits. However, EEG has not been widely utilized during anesthesia and surgery.<sup>4</sup> Since the voltages recorded from the scalp are minute, they are easily lost in the noisy electrical atmosphere of the operating room (e.g. electrocautery). The recordings are voluminous and do not readily lend themselves to interpretation for trend recording. In many cases a special technician is required to operate equipment and collect EEG data.

Recently, we have utilized a Cerebral Function Monitor<sup>®</sup> (CFM)\* to assess cerebral activity during open heart surgery. In this system, an amplified EEG signal obtained from two electrodes placed over both parietal regions (plus a neutral ground electrode) is passed through a wide band filter that rejects frequencies below 2 Hz and those above 15 Hz, thus, minimizing artifacts and electrical interference.<sup>5,6,7</sup> Following logarithmic compression and

peak to peak rectification, the output (micro-volts) is (essentially a straight line drawn through the peaks of the compressed signal) then displayed on a write-out recorder (6 or 30cm/hr). The lower edge of the trace (height above the baseline) indicates the actual level of activity. The variability in EEG amplitude is seen in the width of the trace. The cerebral activity is recorded as part of a four channel write-out recording (Figure 1). Key to the system for long term trend recording is the ability to measure electrode impedance and thus, insure adequate conductivity and eliminate artifact. A study was undertaken to assess the value of such monitoring during open heart surgery.

#### METHODS AND MATERIALS

Fifty patients with congenital and acquired heart disease (ages 2 months to 64 years) undergoing cardiac surgery were evaluated (Table I).

TABLE I

PROCEDURE	NO. PATIENTS
Coronary Artery Surgery	31
Coronary Artery Surgery and Valve Replacement	7
Valve Replacement	5
Correction of Congenital Heart Defect	5
Left Ventricular Aneurysmectomy	2
	50

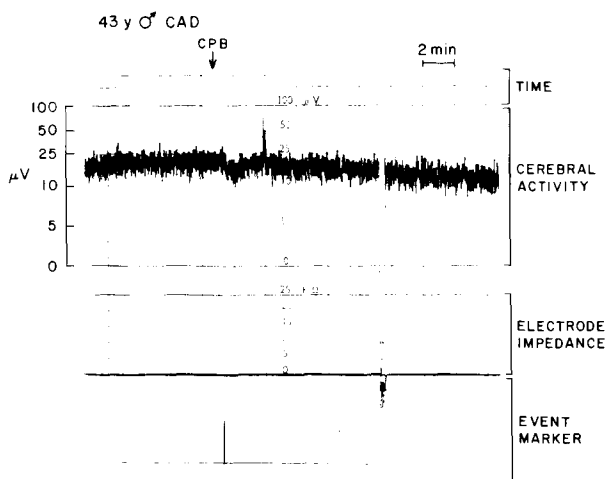


Figure 1. A typical 4-channel recording showing a time marker, cerebral activity channel, electrical impedance channel and an event marker. This trace of a 43 year old male with coronary artery disease (CAD) undergoing coronary artery bypass graft surgery demonstrates no change in cerebral activity at the onset of CPB.

Intramuscular premedication consisted of secobarbital (4mg/kg) and morphine (0.1 mg/kg) (children) or diazepam (0.15mg/kg.) and morphine (0.15mg/kg) (adults) one hour prior to the induction of general anesthesia. In children, anesthesia was maintained with nitrous oxide and halothane. In adults, anesthesia was maintained with nitrous oxide, halothane and pancuronium. The premedication and anesthesia were obviously adjusted to the patient's physiologic status:

Following induction of general anesthesia, and prior to cardiopulmonary bypass, the CFM electrodes were placed and cerebral activity recorded (30cm/hr) until the conclusion of surgery. After CPB is initiated, mean arterial blood pressure is maintained by regulation of flow from the oxygenator and peripheral vascular resistance of the patient. Peripheral constriction is controlled by a vasopressor (phenylephrine) or anesthetic vasodilator (halothane) which also served as the maintenance anesthetic while on cardiopulmonary bypass. Four types of oxygenators were employed: Temptrol® (Bentley), Veriflo® (Travenol), H-1000 Bubble Oxygenator® (Harvey) and Kolobow® (Sci-Med). For this study the ideal pump flow was calculated as 2.2L/min/m<sup>2</sup> and the mean blood pressure was 60-100mmHg. In all cases, a 40  $\mu$  micro-emboli filter was used in the arterial circuit. Hemodilution (hematocrit 22-28%) was employed in all but 2 cases. Temperature during cardiopulmonary bypass was decreased to 28°C in 36 patients (1 patient to 26°C). Two children underwent surface induced profound hypothermia with circulatory arrest.<sup>8</sup> Arterial oxygen tension was maintained above 150mmHg and PaCO<sub>2</sub>  $\approx$  30-35mmHg. Monitoring, in addition to the CFM, consisted of electrocardiogram, arterial, pulmonary artery, and/or left atrial pressures, cardiac output (thermodilution), esophageal and rectal temperatures, blood gases (arterial and venous), serum potassium, and urinary output.

## RESULTS

Prior to CPB, no changes were noted in cerebral activity. However, immediately following onset of CPB, 3 distinct patterns emerged. An increase in cerebral activity (>5 micro-volts) was observed in 10% of patients (5/50) (Figure 2). A decrease in electrical activity (>5 micro-volts) was noted in 28% (14/50). No initial changes were observed in 52% (31/50). However in the latter category, a secondary decrease was noted in 75% (24/32) which was related to a decrease in temperature (Figure 3) (see discussion). At the conclusion of surgery, cerebral activity returned towards normal. The units of change in cerebral activity are measured as micro-volt seconds (decrease or increase [ $\mu$ V  $\times$  seconds]). Other than specific techniques of temperature reduction and surface induced profound hypothermia decreases range from 100 $\mu$ V seconds to 1850 $\mu$ V seconds. In this series, two patients were noted to have clinical cerebral impairment post-operatively. One sustained a mild psychosis several days post-operatively. The cerebral function record showed no change in activity. The other patient suffered multiple strokes during the peri-operative period and expired 17 days following surgery. This patient's cerebral function trace revealed a prolonged 6 V

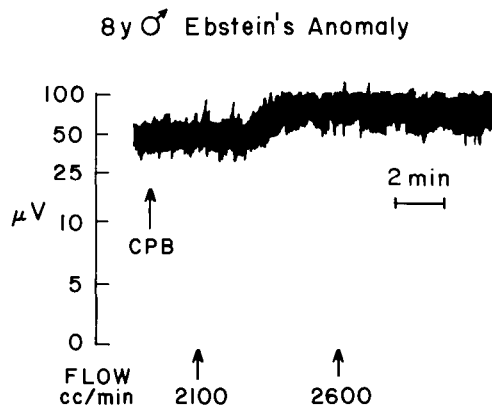


Figure 2. An 8 year old male with Ebstein's anomaly showing 12.5 micro-volt rise with increased perfusion flows during CPB.

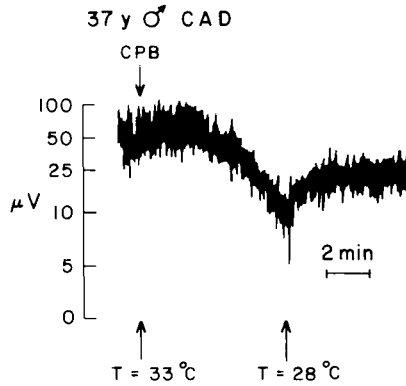


Figure 3. A 37 year old male undergoing coronary artery surgery showing a typical transient decline in cerebral activity associated with a decrease in temperature to 28°C.

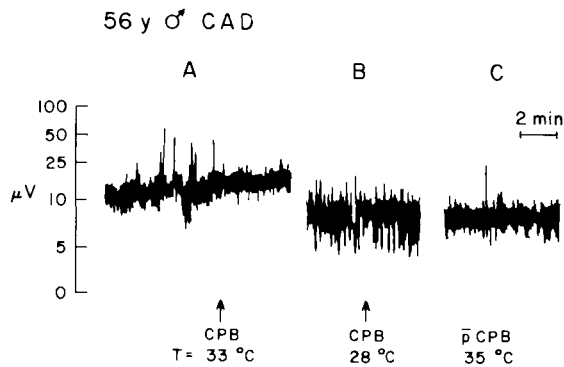


Figure 4. A 56 year old male undergoing coronary artery surgery who had neurologic dysfunction post-operatively. Normal activity prior to CPB (A). During CPB, at 28°C, the patient developed a burst suppression pattern with a very low level of activity (4μV) (B). Following CPB, the patient's cerebral activity has not been restored to normal (C).

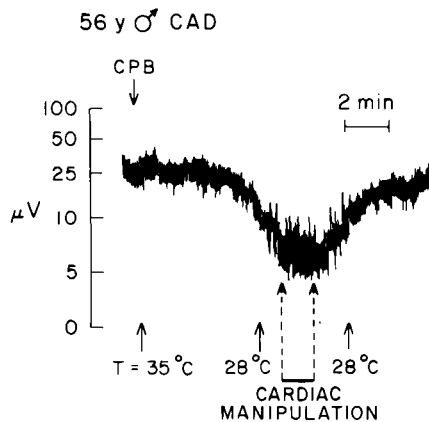


Figure 5. A 56 year old male undergoing coronary artery surgery showing the expected decrease in electrical activity at 28°C. However, with cardiac manipulation a further decrease was observed which returned to baseline levels following cessation of this maneuver.

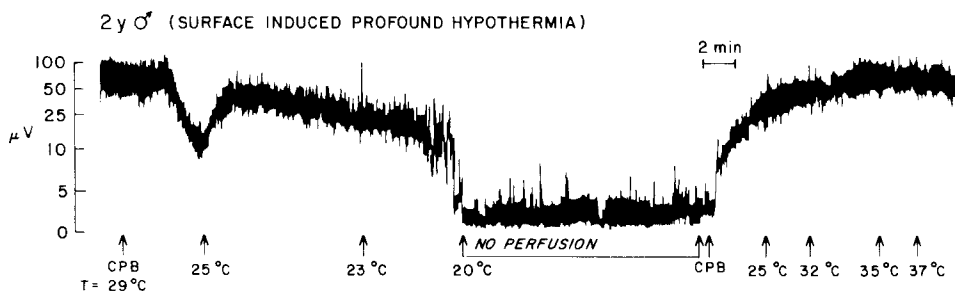


Figure 6. A 2 year old male for repair of the ventricular septal defect (VSD) using surface induced profound hypothermia. Note the marked decrease in activity at 25°C. During this protocol, there is a period of 15 minutes of circulatory arrest (no perfusion) at 20°C during which the VSD is closed. Note virtually, no cerebral activity at this point. When cardiopulmonary bypass is reinstated there is a marked increase in cerebral activity which is progressive as temperature is restored at 37°C.

decrease with "burst suppression patterns" (indicating cerebral depression). At the conclusion of surgery, he still had a marked depression of cerebral activity (7-8 $\mu$ V) (Figure 4).

## DISCUSSION

The reported incidence of neurologic sequelae following cardiac surgery is quite variable. While Javid estimated the incidence as 53%, Branthwaite observed that 10/140 patients suffered neurologic dysfunction following open heart surgery<sup>1,9</sup>. However, 62.9% of all his patients had a significant alteration in cerebral activity during CPB. Stockard noted an excellent correlation between changes and hypotension (<50mmHg) during CPB.<sup>2</sup> When measures are taken to diagnose these problems, the incidence can be reduced.<sup>1,2,3</sup> Early in this series, we observed that perfusion pressure at the onset of CPB significantly affected the level of cerebral activity. Even transient perfusion pressures below 50mmHg were associated with marked decreases in cerebral activity. However, when cerebral function monitoring was added to our protocol, perfusion techniques were altered to assure that arterial pressure did not fall below 50mmHg. Thus, most records were similar to Figure 1.

In 5 cases, an increase in activity was noted (Figure 2). We hypothesize a cardiac output may have been reduced either secondary to the intrinsic heart disease or the cardiac depressant effects of anesthesia. With the initiation of cardiopulmonary bypass, flow was increased above this minimal level. Alternatively, at the onset of CPB, there are marked changes in the concentration of anesthetic agents which may account for the patient being at a very "light" level of general anesthesia. Occasionally, this CFM trace was the only sign of poor cerebral perfusion. In one case, when the heart was manipulated during CPB, an unexpected drop in cerebral activity was observed which was promptly restored by having the surgeon cease manipulation (Figure 5).

In our series, a decrease in cerebral activity was deemed an appropriate physiologic response to a decrease in temperature (<28°C) (Figure 3) or elective circulatory arrest cessation of perfusion (Figure 6).

There are disadvantages associated with the use of the CFM. The monitor is not designed to detect focal cerebral disturbances. Since the sensitivity of the instrument is limited, the nature, extent and location of injury cannot be easily determined. Most anesthetic drugs do not appear to alter the CFM trace.<sup>10</sup> Levels of cerebral activity which are

pathologic may occur beyond the filter range of the CFM. Although the CFM trace may indicate considerable cerebral dysfunction, clinical neurologic sequelae do not inevitably result.<sup>1,5</sup>

In a daily clinical use, the Cerebral Function Monitor can serve as a first line diagnostic device. When cerebral impairment is suspected, further evaluation should be undertaken both intra- and post-operatively.

\*Devices Limited, New Brunswick, New Jersey 08903

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